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Cardiovascular, metabolic and fetal brain function observation following total cord occlusion

Wolfgang Künzel, Leon I. Mann, Amrutha Bhakthavathsalan, Jahagir Airomlooi

From the Department of Obstetrics and Gynecology, University of Vermont, College of Medicine, Burlington, Vermont

and the

Universitäts-Frauenklinik, Würzburg

The cardiovascular response to total cord occlusion (TCO) results in fetal bradycardia [1]. This deceleration in fetal heart rate is generated by vagal stimulation and acts as an early indicator of cardiovascular disturbances during labor.
The present knowledge of the patho-physiological response of the fetal heart rate to TCO is however still inconclusive [2,5,13,14]. The factors which are involved in the rise of the fetal blood pressure are not well established and so is the quantitative relationship between the rise of the fetal blood pressure and the fall of heart rate. In addition no experiments were performed which show the influence of TCO on quantitative alterations of the fetal oxygenation and fetal brain function.
The present study was performed to investigate with an experimental model in sheep these questions under physiological and also under pathological conditions. The pattern of fetal heart rate in threatened fetal situation should be known for clinical reasons. The fetal brain function was investigated in terms of fetal EEG.
It could be demonstrated that fetal heart rate fell as a result of the arterial blood pressure elevation. However the elevation of the fetal blood pressure was dependend on the blood pressure and the umbilical blood flow before occlusion of the umbilical cord. The fetal EEG showed a dramatic response after 60–80 sec with a complete disappearance of the brain electrical activity.

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1 Methods

1.1 Material, anesthesia and surgical procedure

The experiments were performed on 11 sheep with a dated gestational age of 126 to 137 days. Anesthesia was induced with 3–5% halothane and 30% oxygen in nitrous oxyd by means of a mask. The ewe was then intubated and after relaxation mechanically ventilated. Anesthesia was maintained with 0.5–1 % halothane, 30 % oxygen and N_2O. The abdomen was opened by a midline incision

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and one uterine horn exposed. An incision was made in an avascular area of the uterine horn and the fetus exteriorised. Two catheters were placed via the left femoral artery of the fetus (FA) into the fetal aorta, one used for continous monitoring and the other for blood sampling. The common umbilical vein was exposed by an abdominal incision superior of the umbilicus. An electromagnetic flow probe with an inner diameter of 5–7 mm was then placed around the vein. An inflatable cuff was placed around the umbilical cord. For fetal EEG monitoring the fetal scalp was incised. Two extradural electrodes were placed in burr holes made with a dental drill over the right hemisphere.

1.2 Experimental procedure and measurements

Total cord occlusion (TCO) was performed 27 times in 11 animals: in 13 cases for at least 10 sec and in 5 of them for a maximum of 300 sec. In 21 cases the mean arterial blood pressure, fetal heart rate and umbilical blood flow could be measured simultanously. Fetal arterial blood (FA) was drawn from one aortic catheter and analysed for oxygen saturation (SO_2) (IL/Co-oxymeter), pH and P_{CO_2} (mmHg) (IL-gasanalyzer). The blood pressure – systolic and diastolic BP – was measured with strain gauge transducers (STATHAM Pb 23) and fetal heart rate (FHR) by means of a cardio-tachometer (BECKMAN-Instruments) which was triggered by the blood pressure wave. Umbilical blood flow was measured with an electromagnetic flowmeter (STATHAM PS 2002). All parameters were continously recorded throughout each experiment with an Beckman-8-channel dynograph.

A second dynograph was used for FEEG monitoring (paper speed 2.5 cm/sec).

2 Results

2.1 Fetal oxygenation, metabolic and brain function observation following total cord occlusion

2.1.1 Fetal oxygen saturation (SO_2)

The interruption of the umbilical circulation was paralled by a discontinuation of the oxygen supply

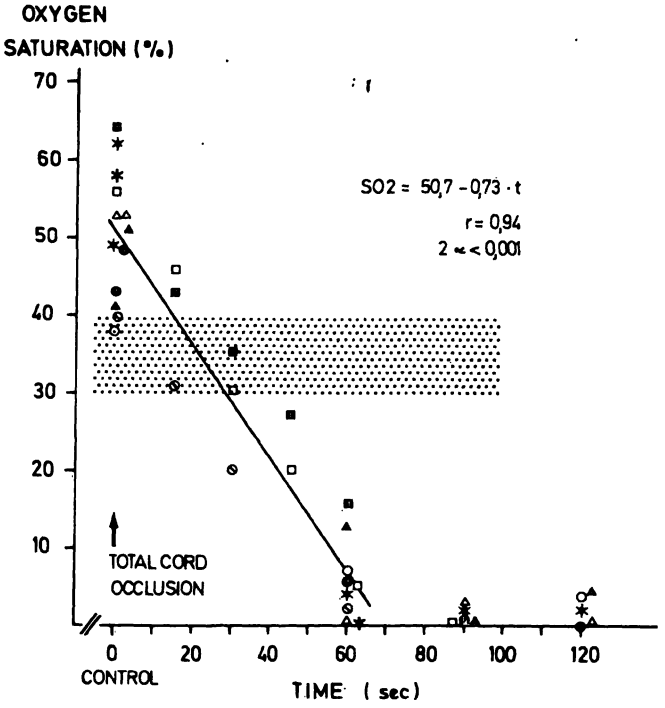


Fig. 1. Relationship between the oxygen saturation in the fetal arterial blood and the time following total cord occlusion. A linear regression was calculated for the data measured over 60 sec. After 30 sec about half of the oxygen was consumed and after 69 sec the SO_2 is zero. The dotted area shows the critical range of fetal SO_2 were the oxygen consumption of the fetus starts to fall.

from the mother to the fetus and resulted in an exhaustion of the fetal oxygen stores. Fig. 1 shows the oxygen saturation in the fetal arterial blood. Each symbol indicates one animal in one or two experiments. In three cases (open squares, filled squares, circle with dash) blood was drawn in 15 sec intervals. The regression line was calculated for the first 60 sec. Independently on the oxygen saturation at control, there was a linear fall of the FA SO_2. After 30 sec about half of the fetal oxygen was consumed and after 60 sec the FA SO_2 of the fetal arterial blood was lower than 20%. After 80 sec however the oxygen stores of the fetus have been completely exhausted.

2.1.2 Metabolic alteration in the fetal arterial blood

Fig. 2 shows in a semilogarithmic plot the relationship between the P_{CO_2} and the pH of the arterial blood of the fetus. The solid line is the theoretical relationship between the P_{CO_2} and pH if the base

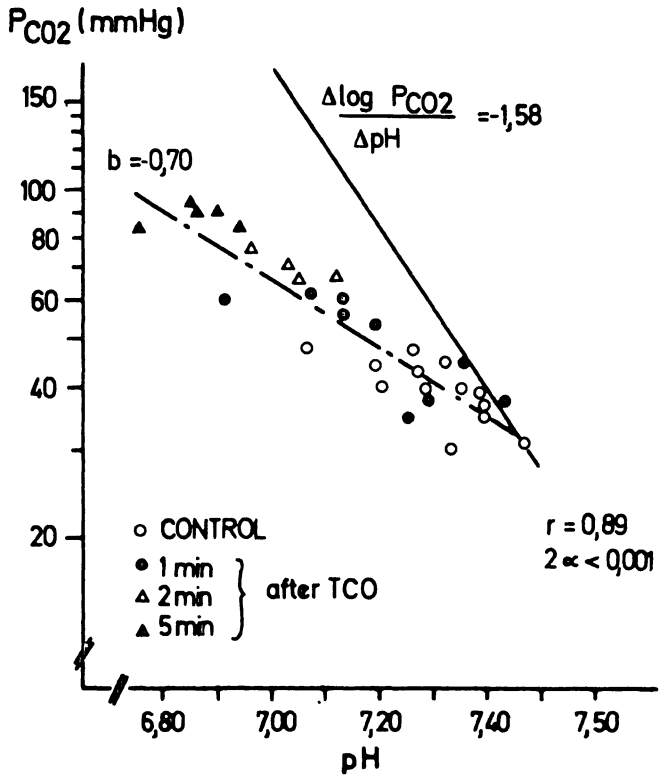


Fig. 2. The P_{CO_2} on a logarithmic scale is related to the pH according to SIGGAARD-ANDERSON-Nomogram (1962). The solid line is the relationship between P_{CO_2} and pH if no alterations in buffer base take place (regression coefficient – 1.58). The dotted line is the calculated regression for the measured data. Both lines deviate significantly from each other. The regression coefficent is –0.70. The different symbols indicate the time following total cord occlusion (TCO).

excess of the blood is not altered (SIGGAARD-ANDERSON 1965 for human adult blood $\Delta\log P_{CO_2}/\Delta pH = -1.58$).

The dashed line is the calculated regression line for the P_{CO_2}-pH relationship in sheep fetus at control (open circles), after 1 min (filled circles), after 2 min (open triangles) and after 5 min (filled triangles) of total cord occlusion.

During the control period a good relationship between the pH and the PCO_2 could be established demonstrating that with increasing PCO_2 the pH fell. The same exists for the 1 min period had became clearly evident for the 2 and 5 min compression time. If we assume that the sheep fetal blood has about the same buffer properties as the human blood the caculated regression coefficient of –0.7 deviates significantly from the theoretical relationship of –1.58. This fact demonstrates that during cord compression the buffer base fell.

2.1.3 Fetal brain function observation

In 5 fetuses the fetal EEG was measured throughout total cord occlusion. In all cases a typical recording was seen as shown in Fig. 3. 40–50 sec following total cord occlusion the EEG showed a decrease in amplitude and a drop out of faster frequencies and after 80 sec no signs of electrical activity could be seen anymore.

2.2 Fetal cardiovascular alterations following total cord occlusion

2.2.1 Fetal heart rate (FHR) and arterial blood pressure (BP)

The abrupt interruption of the umbilical circulation was followed by an increase of the systolic and diastolic blood pressure accompanied by a fall of FHR (Fig. 4).

The systolic and diastolic blood pressure was at control 53 (SD 7) (N = 9) mmHg and 39 (SD 5) (N = 9) mmHg, respectively. There was a continous increase up to 60 and 90 sec: systolic BP 86 (SD 19) (N = 9) and 106 (SD 5) (N = 5) mmHg; diastolic BP 59 SD 7) (N = 9) and 63 (SD 4) (N = 5) mmHg, respectively following TCO. The increase was associated with a rise in pulse pressure.

Fetal heart rate fell within 29 sec from 174 (SD 36) b/min to about 80 b/min and remained there for the time of compression.

2 2.2 The analysis of the blood pressure and the fetal heart rate response following total cord occlusion during the first time sequence of 10 sec

The response of the fetal blood pressure and fetal heart rate was analysed during the initial time sequence of 10 sec following total cord occlusion in 27 observations. Different experimental conditions were present i.e. it was possible to study fetal heart rate and blood pressure response at normal and pathological fetal blood pH, SO_2 and umbilical blood flow, respectively. The results were grouped according to the pH in the fetal arterial blood and considered as normal if the pH was 7.31 and more

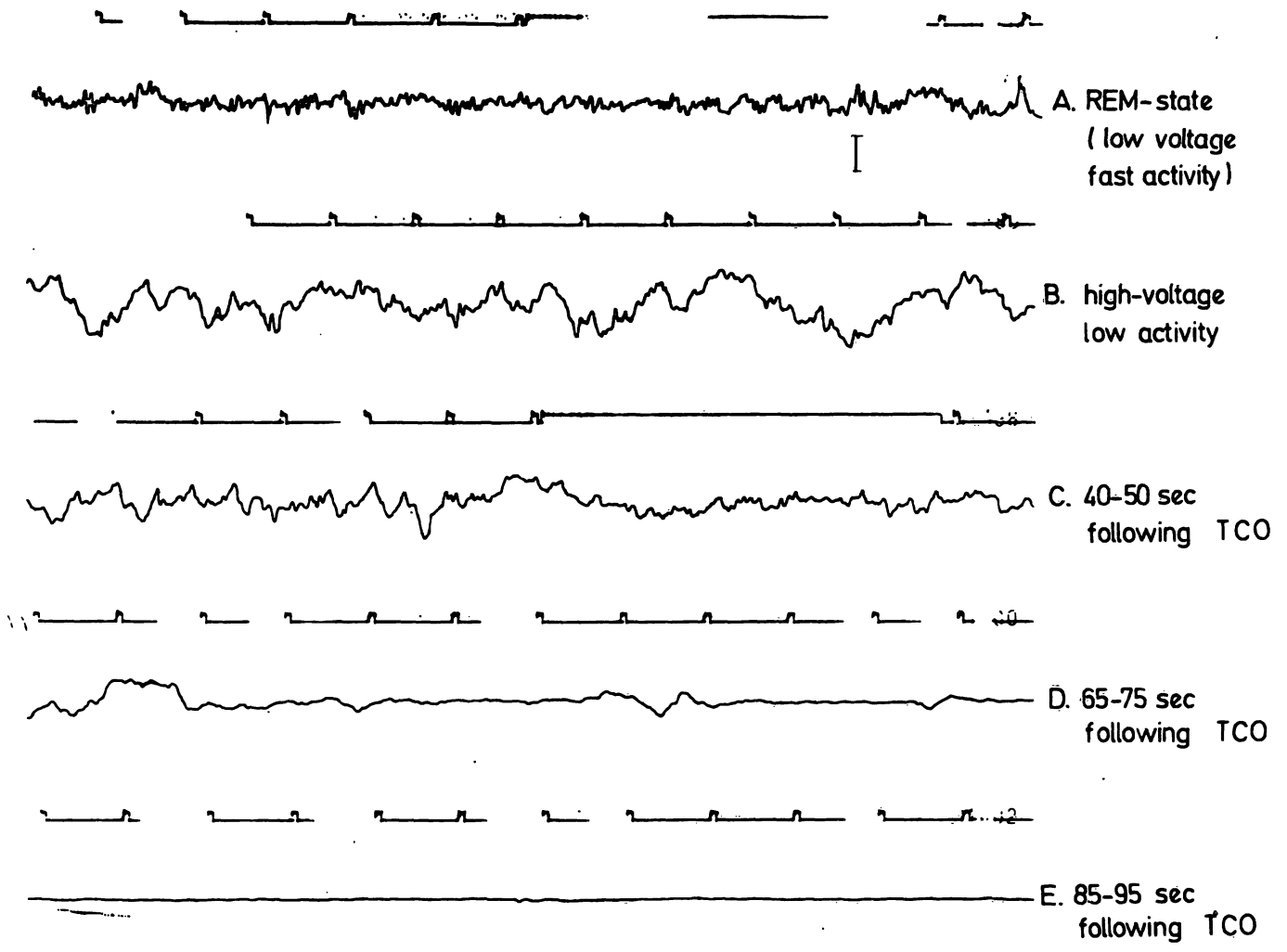


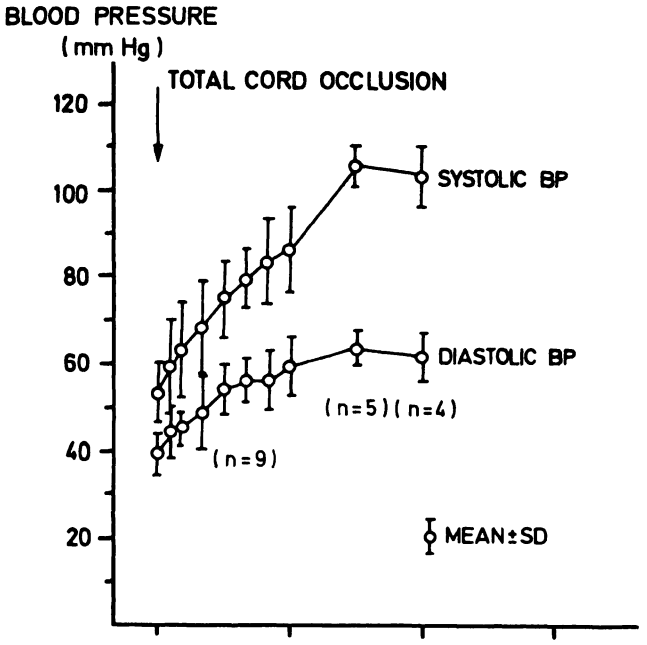
Fig. 3. Fetal EEG before and following total cord occlusion (TCO). A and B shows the fetal EEG at control with low voltage-fast activity and high voltage-low activity, respectively. The time marking is 1 sec, the vertical bare in A indicates 100 μV. Following TCO a flat EEG is achieved after 60 sec, a time were the FA SO_2 approaches zero.

(N = 12). Moderate acidosis was assumed if the pH was 7.21–7.30 and severe acidosis was assumed to be present if the pH was 7.20 and less (Tab. I).

2.2.3 Fetal systolic and diastolic blood pressure and fetal heart rate.

In Fig. 5 the systolic and diastolic blood pressure of the fetus of 27 experiments during the first 10 sec following total cord occlusion is shown. According to Tab. I the open circles show values where the fetus is in physiological conditions, the filled circles are values were the fetus had a moderate acidoses and the triangles are fetus with severe acidosis.

There was a close relationship between the blood pressure at control and the pH of the fetus ($\overline{p}$ = $-488 + 72.6 \cdot$ pH, N = 27, r = 0.79, $2a < 0.001$). The rise of the systolic as well as the diastolic blood pressure was related to the metabolic status of the fetus. The blood pressure achieved its maximum value after 2–3 sec following total cord occlusion. After a short tendency to fall, evident in the group with normal pH, the blood pressure rose again after about 8–10 sec. The fetal heart rate showed a continous fall from about 170 b/min to about 120 b/min after 10 sec. The fall of the fetal heart rate showed no significant difference between these groups.



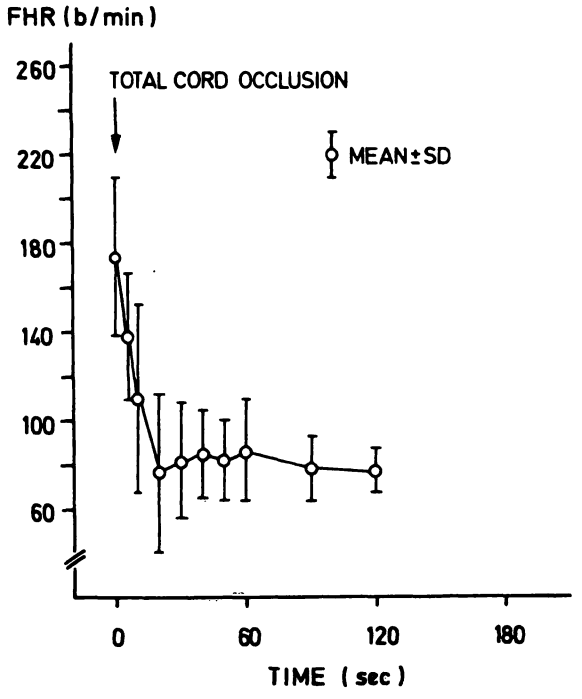


Fig. 4. Systolic (S) and diastolic (D) blood pressure (BP), and fetal heart rate (FHR) following total cord occlusion (TCO). With total cord occlusion the blood pressure increased and fetal heart rate fell. The measurements are given as mean ± standard deviation. A higher time resolution of the alterations in fetal blood pressure of the first 10 sec following TCO is shown in Fig. 5.

Tab. I. The pH, base excess and the oxygen saturation in the fetal arterial blood and umbilical blood flow before total cord occlusion. The values were grouped according to the pH measured before the occlusion and related to the cardiovascular observations as shown in the figure 5 ($\bar{x}$ = mean, SD = standard deviation, N = number of observations).

	pH	base excess (meq/l)	oxygen saturation (%)	umbilical blood flow (ml/kg/min)
	$\bar{x}$ SD (N)	$\bar{x}$ SD (N)	$\bar{x}$ SD (N)	$\bar{x}$ SD (N)
○ normal acid-base status (pH > 7.30)	7.37 0.04 (12)	− 3.9 1.8 (12)	60.7 9.9 (12)	148 79 (10)
● moderate acidosis (pH 7.21 − 7.30)	7.27 0.01 (8)	− 6.9 0.5 (8)	54.3 6.2 (7)	88 9 (5)
△ severe acidosis (pH ⩽ 7.20)	7.18 0.02 (7)	−13.6 1.6 (7)	25.9 12.9 (7)	5 8 (6)

2.2.4 The mean arterial blood pressure after 3 sec following total cord occlusion and its relationship to the blood pressure, umbilical blood flow before TCO and to the fetal heart rate following TCO

An attempt was made to analyse the relationship of the blood pressure and the umbilical blood flow at control and the rise of the fetal blood pressure following total cord occlusion. In addition it was important to know how the rise of the blood pressure was counteracted by slowing of fetal heart rate. The measured values 3 sec following total cord occlusion were therefore selected. The cardiovascular parameters measured at this time are not a result of the desoxygenation of the fetal blood. They demonstrate most likely the response of the baroreceptors which are simply stimulated by the increase of the blood pressure resulting from the sudden elevation of the umbilical vascular resistance (see discussion).

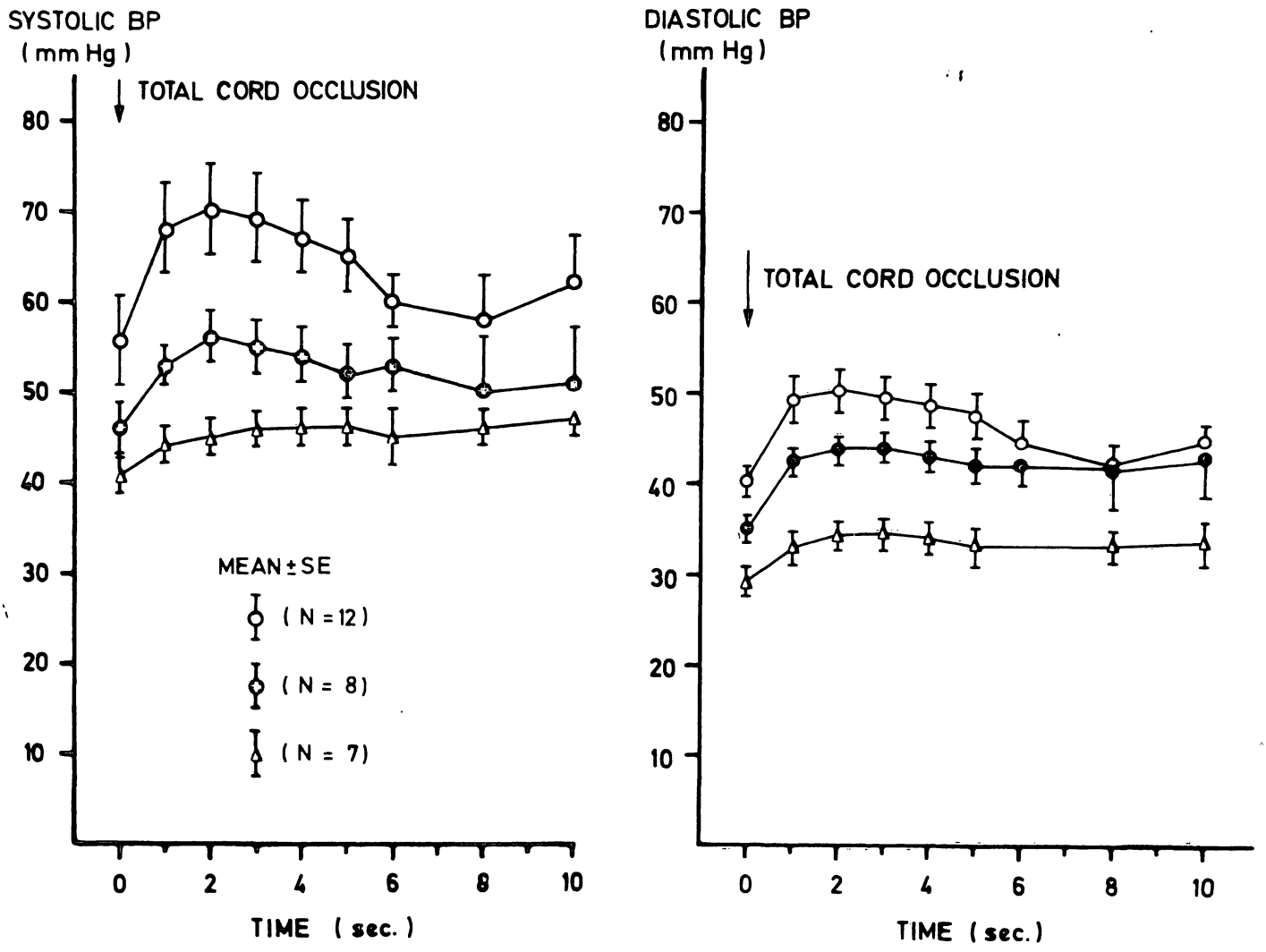


Fig. 5. The systolic and diastolic blood pressure (BP) following total cord occlusion. According to table 1 the different symbols show the response of the fetal BP to TCO in a group with a pH of 7.31 and more (open circles), pH 7.21–7.30 (closed circles) and 7.20 and less (triangles). The values are given as mean ± standard error.
From this figure it is apparent that the BP at control is related to the pH of the fetus and that the increase of the BP following TCO is also related to the status of the fetus.

2.2.5 Mean arterial blood pressure (BP).

Fig. 6 shows the relationship between the rise of the mean arterial BP and the BP at control. At normal fetal BP of about 50 mmHg the BP increased by about 15 mmHg. The BP rose only by about 5 mmHg if the BP at control was 30 mmHg.

2.2.6 Umbilical blood flow (UBF). The increase of the blood pressure was related to the umbilical blood flow at control (Fig. 7). To the respective values a logarithmic regression line was adapted. In the range of an UBF between 40 and 140 ml/kg/min the rise of the arterial BP is roughly proportional to the UBF. If the blood was more than 200 ml/kg/min the increase of the arterial BP exceeds never 20 mmHg.

2.2.7 Fetal heart rate (FHR). In Fig. 8 the fall of FHR 3 sec following total cord occlusion is related to the increase of the mean arterial BP. There exist a linear relationship between both parameters. The regression line runs through zero. With an elevation of the arterial BP of 15 mmHg the FHR fell by about 60 b/min. If the BP increased only by 5 mmHg a drop of FHR by 20 b/min was observed. This indicates that the

fall of FHR was related to the condition of the fetus. It was less pronounced if the fetus had a severe acidosis.

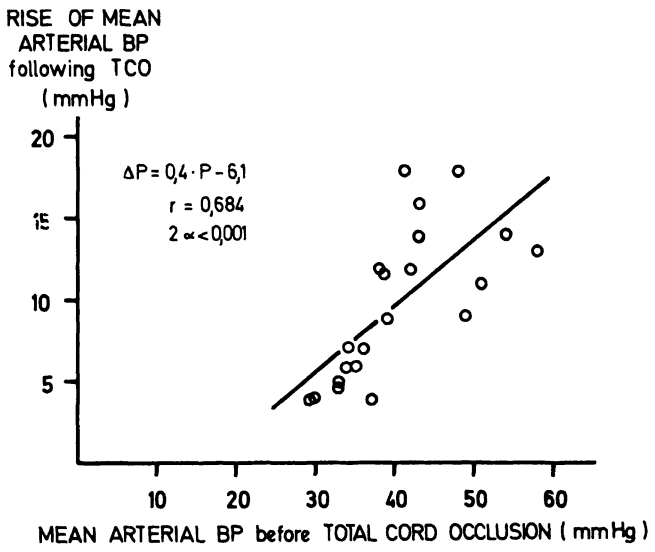


Fig. 6. The relationship between the rise of the mean arterial BP after 3 sec following TCO and the mean arterial BP before occlusion. The increase of the BP following TCO should be twice as high as at control if no alterations of the fetal cardiac output take place, the fetal tissue resistance remains constant and if there is an even distribution of the CO to the fetal tissue and to the placenta. The increase of the BP was however only 20–30% of control. For further explanation see text.

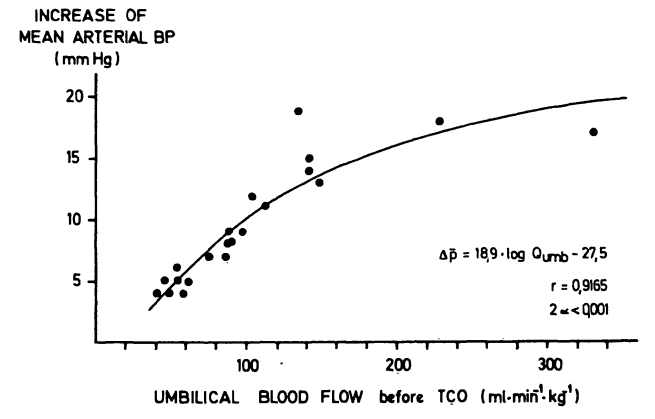


Fig. 7. The increase of the mean arterial blood pressure (BP) measured 3 sec following total cord occlusion related to umbilical blood flow (Qumb) before TCO. A logarithmic regression line was adapted to the respective data. The increase of the BP is almost proportional to Qumb in a range of 40–140 ml/kg/min, however levels off if Qumb is greater than 150 ml/kg/min.

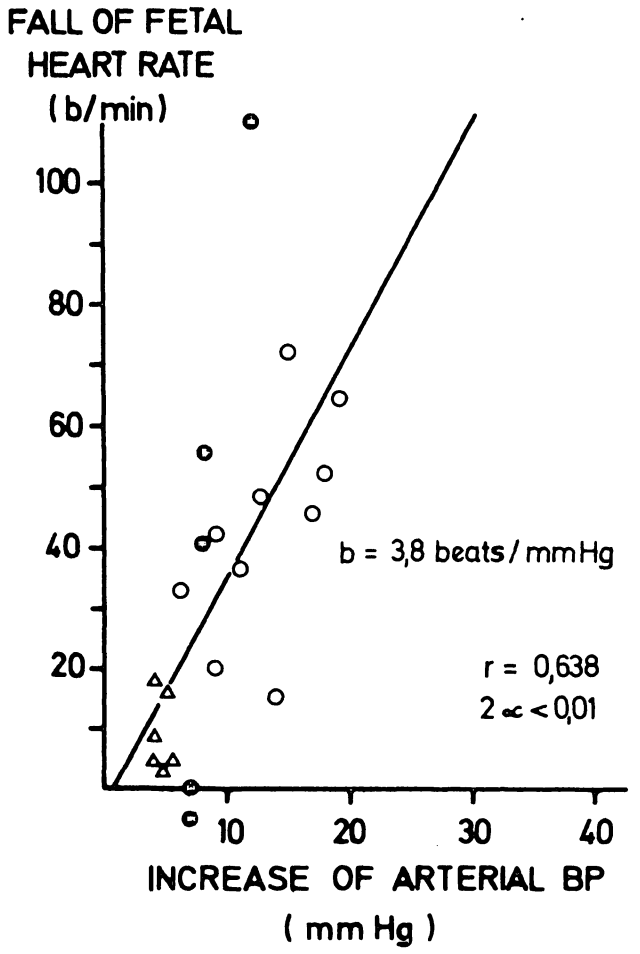


Fig. 8. The fall of fetal heart rate (FHR) estimated 3 sec following total cord occlusion related to the rise of the arterial blood pressure (BP). The different symbols represent the groups shown in table 1. The FHR falls by 3.8 beats/min if the BP is increased by 1 mmHg.

3 Discussion

According to clinical observations the occlusion of the umbilical cord during parturition is the most frequent cause for the fetal heart rate deceleration observed during the last stage of labor [3,8]. The stimulation of the baroreceptors and fetal hypoxia are in this context the mechanisms which are involved, in fetal heart rate slowing as already shown by BARCROFT [1], REYNOLDS [9] for the fetal lamb, YEH et al. [14] for the baboon and from LEE and HON [5] for the human.

Two question have been of major importance: what is the effect of the total cord occlusion on fetal oxygenation and fetal EEG in terms of the time sequence, and secondly it was of interest to

differentiate between the heart rate slowing which is due to barcreceptor stimulation and due to hypoxia as an impact of the chemoreceptors and its relationship to the fetal condition.

3.1 Oxygen supply and fetal EEG following total cord occlusion

The O_2-uptake of the sheep fetus obtained under acute conditions was found to be 6.1 ml/kg/min [4]. Since in total cord occlusion the O_2 transfer from the mother to the fetus is immediately interrupted, the fetus has to use its oxygen stores to meet the requirements of its energy need. The only stores which are available are the oxygen that is bound to the hemoglobin and the oxygen that is dissolved in the fetal blood. As the small amount of oxygen which is dissolved in the fetal blood is of less significance the decline of the oxygen saturation following total cord occlusion can be easily calculated from knowing the hemoglobin concentration (C_{Hb} = 14 g%), the oxygen saturation before occlusion (SO_2 = 50%) and the blood volume of the fetus (80 ml/kg).

If we assume that the need of energy during total cord occlusion is fairly constant and the oxygen is consumed in a constant rate of 6.1 ml/kg/min it can be calculated from (1)

$$t = \frac{1}{V_{O_2}} \cdot BV \cdot SO_2 \cdot C_{Hb} \cdot 1.34 \cdot 6.10^{-3} \text{ (sec)} \quad (1)$$

that the total amount of oxygen which was available at the moment of occlusion is consumed after 74 sec.

This calculated figure is in a good agreement with the decline of oxygen saturation in the fetal arterial blood measured during total cord occlusion (Fig. 1). According to the calculated regression line the fetal SO_2 approaches zero after 69 sec.

The depletion of the fetal oxygen stores was paralled by an increase of the fetal P_{CO_2} and a fall of the pH in the fetal blood (Fig. 2). If the buffer base would be constant the regression coefficient for the $\Delta\log P_{CO_2}/\Delta pH$ is −1.58. In the present observations however the calculated regression line deviated significantly from this theoretical relationship: $\Delta\log P_{CO_2}/\Delta pH$ = −0.70. This indicates that even at short episodes of 3–5 min hypoxia the buffer base of the fetal blood fell.

The observations of the fetal EEG are especially of clinical significance. In many cases heart rate decelerations during vigorous contractions occure for 60 sec and last even longer. So it was also surprising to see that **the changes in the electrical activity of the fetal brain occured almost after 45 sec of total cord occlusion (Fig. 3). The first alterations of the fetal EEG took place after a critical O_2-saturation of about 15% had been achieved.** This is in good agreement with previous observations [6]. After 60 sec the EEG became flat and remained so for the time of occlusion. **At present no data are available concerning the longterm effect of the intermittently occuring isoelectric EEG following TCO.** It may be speculated that even short episodes of hypoxic insults have an impact on the brain cells which can not be revealed by the applied method of EEG monitoring during the recovery period. One should keep that in mind observing variable heart rate pattern during parturition.

3.2 The cardiovascular response to total cord occlusion

The cardiovascular response to total cord occlusion has to be distinguished from a response which is due to the stimulation of the baroreceptors resulting from the elevated resistance in the umbilical circulation and a response which is due to hypoxia. The fetal circulation time from the umbilical vein to the carotid artery was 1.9 ± 0.2 sec [7]. The first response to total cord occlusion however took place within 0.25 sec, so that **we may assume that the initial cardiovascular response was due to a baroreceptor stimulation.** Fetal blood sampling in short intervall of 5 sec following fetal aorta occlusion reveal also that the oxygen saturation is not affected within 5 sec [11], so that chemoreceptor stimulation may not interfer with the measurement conducted 2–3 sec following total cord occlusion.

The initial cardiovascular response to total cord occlusion was a rise in fetal blood pressure and a fall in fetal heart rate. These cardiovascular alterations are consistent with the findings of others

[1,2,9,13,14]. The initial response may be explained by the sudden redistribution of the placental fraction of the cardiac output following TCO to the blood flow that perfuses the fetal tissue. This response is similar to that described by SHINEBOURNE et al. [11] occluding the fetal aorta in conscious sheep throughout gestation.

Theoretically this increase in fetal blood pressure should be twice as high as before occlusion. However umbilical blood flow, fetal blood pressure and finally fetal heart rate, which are related to the fetal condition are also involved in the rise of fetal blood pressure following TCO.

According to equation (2) the fetal arterial blood pressure ($\bar{p}a$) is dependent on the fetal cardiac output (C.O.) and the flow resistance (R) which is offered to C.O. by the fetal tissue and the placenta.

$$\bar{p}a = C.O. \cdot R \qquad (2)$$

Under physiological conditions 60 % of the fetal C.O. are directed to the placenta and 40% to the fetal tissue [10]. Fetal blood pressure is so far related to both tissue (Q_T) and placental blood flow (Q_P) and tissue (R_T) and placental resistance (R_p) · R_T and R_P are in parallel and so it follows (3)

$$\bar{p}a = (Q_P + Q_T) \cdot \frac{R_P \cdot R_T}{R_P + R_T} \qquad (3)$$

According to this equation and to the above mentioned distribution of C.O. the $\bar{p}a$ would increase by 70% if the cord is totally occluded i.e. by an increase of the umbilical resistance to infinit. However this increase of the blood pressure of 70% would only occure, if the C.O., the force of cardiac contraction and the resistance to blood flow in the fetal tissue remain constant during the time of occlusion.

It is most likely that this does not take place. The relationship between the blood pressure at control and the blood pressure after 3 sec indicates an increase of 20–30%. The distribution of blood flow to the placenta and the fetal organs before occlusion may be related too, as the fall of fetal heart rate is also responsible for the reduced blood pressure elevation following total cord occlusion.

The rise of the blood pressure is exspected to be high if the umbilical blood flow is normal and low if the blood flow approaches zero. This may also explain the drop of fetal heart rate which is more marked under physiological conditions.

Quantitative assessment of baroreflex activity has not been carried out during total cord occlusion. The only data available are gained from the experimental set up used by SHINEBOURNE et al. [11] which is quite similare to our study. With an inflatable balloon which was placed in the fetal aorta the baroreflex activity in conscious sheep throughout gestation was studied. The proportion of positive baroreflex responses increased with advancing gestational age. For the estimation of the reflex activity a different approach is possible and given in detail in their paper. In the present findings the increase in blood pressure and the fall of fetal heart rate was not always constant, so that we have selected a constant time of 3 sec after total cord occlusion was started for analyzing. The data reveal a baroreceptor response which is fairly invariable of 3.8 b/min and mmHg of fetal mean arterial blood pressure elevation and which was not related to the condition of the fetus.

In summary it may be stated that the first response of fetal heart rate deceleration which takes place following total cord occlusion is a baroreceptor response due to the increased vascular resistance in the umbilical circulation lasting for at least 15–20 sec. This response is generated by an increase of the fetal blood pressure and might explain in clinical situations the sharp fall of fetal heart rate starting with a contraction of the uterus and its disappearance after relaxation. This short episodes of fetal heart rate deceleration are considered not to do any harm to the fetus if lasting not longer than 15 sec, especially if they arise from a normal baseline fetal heart rate. **Heart rate decelerations of longer duration signal however hypoxic episodes and are of no good prognostic value**, especially if they arise from an elevated baseline fetal heart rate.

Summary

The effect of total cord occlusion (TCO) on the time sequence of fetal oxygenation, metabolic and cardiovascular parameters and on brain function of the sheep fetus was studied.
It was also of importance to differentiate between cardiovascular alteration following TCO due to hypoxia and baroreceptor response, respectively.
The investigations were performed on 11 near term pregnant sheep with dated gestational age of 126–137 days. Catheters were placed in the fetal aorta for monitoring fetal arterial blood pressure (FA BP), fetal heart rate (FHR). Blood samples were drawn and analysed for pH, P_{CO_2} and SO_2. An electromagnetic flow probe was placed around the common umbilical vein. Fetal EEG was measured over the right hemisphere. All parameters were studied over a varying time of TCO.

Results:
Following TCO the FA SO_2 decreased liniarely paralled by a fall of the FA pH, an increase of the P_{CO_2} and succeeded by a metabolic acidosis. The fetal EEG became flat after about 60 sec fetal hypoxia.
Fetal systolic and diastolic blood pressure increased within 0.25 sec accompanied by a fall of FHR.
A closer analyses of the FHR and FA BP response during the initial period following TCO indicates that the rise of FA BP is related to the acid base status of the fetus.
The physiological mechanisms involved in the rise of the FA BP and fall of FHR are however the FA BP and the umbilical blood flow at control, since baroreceptor response was estimated to be constant (3.8 b/mmHg).

Conclusions:
From the observations it is concluded that the initial response of FHR following TCO is a baroreceptor response due to the sudden rise of FA BP. This episode is shortlasting and in case of fetal heart rate interpretation during labor of less importance.
The major impact and responsible for heart rate slowing is the fetal hypoxia leading to the extreme rise of the FA BP and slow down of FHR.

Keywords: Acid base balance, baroreceptor response, fetal blood pressure, fetal EEG, fetal heart rate, total cord occlusion.

Zusammenfassung

Die Beobachtung cardiovaskulärer und metabolischer Parameter sowie der fetalen Gehirnfunktion nach vollständiger Nabelschnurabklemmung.
Es war das Ziel dieser Untersuchung zu bestimmen, mit welchem zeitlichen Abstand Änderungen der fetalen Oxigenierung, der metabolischen und cardiovaskulären Parameter und der Gehirnfunktion in Folge eines totalen Nabelschnurverschlusses beim Schaffeten auftreten.
Dabei wollten wir auch differenzieren zwischen cardiovaskulären Änderungen, die auf die Hypoxie zurückgehen und Änderungen, die durch die Barorezeptoren vermittelt werden.
Die Experimente wurden an 11 trächtigen Schafen am Termin durchgeführt, wobei das Gestationsalter zwischen 126 und 137 Tagen lag. Um den fetalen arteriellen Blutdruck (FA BP) und die fetale Herzfrequenz (FHR) aufzuzeichnen, legten wir Katheter in die fetale Aorta. Zwecks Bestimmung von pH, P_{CO_2} und SO_2 wurden Blutproben entnommen. Um die Nabelvene wurde ein elektromagnetischer Meßfühler gelegt. Das fetale EEG leiteten wir über der rechten Hemisphäre ab. Alle Parameter wurden bei unterschiedlicher Dauer der Nabelschnurabklemmung beobachtet.

Ergebnisse: In Folge des Nabelschnurverschlusses nahm die Sauerstoffsättigung im fetalen arteriellen Blut (FA SO_2) linear ab bei gleichzeitigem Abfall des pH und einem Anstieg des P_{CO_2}. Danach bestand dann eine metabolische Acidose. Das fetale EEG wurde nach einer etwa 60 sec lang bestehenden Hypoxie flach.
Der fetale systolische und diastolische Blutdruck stieg innerhalb von 0.25 sec an und wurde begleitet von einer sinkenden Herzfrequenz.
Eine genauere Analyse der FHR und des FA BP in der Phase unmittelbar nach dem Nabelschnurverschluß ergab, daß der Blutdruckanstieg in Beziehung zu dem Säure-Basen-Status des Feten zu setzen ist.
Die physiologische Reaktion besteht letztlich in dem Anstieg des FA BP und in dem Abfall der FHR, wobei jedoch dem FA BP und der Kontrolle der Nabelschnurdurchblutung besondere Bedeutung zukommt, da die Barorezeptorenantwort konstant mit 3.8 b/mmHg registriert wurde.

Schlußfolgerungen: Wir müssen unsere Beobachtungen so deuten, daß in der Anfangsphase nach der Abklemmung der plötzliche Blutdruckanstieg die Barorezeptoren stimuliert, was seinerseits einen Abfall der FHR zur Folge hat. Diese Phase ist aber von kurzer Dauer und hat für die Beurteilung der FHR während der Wehen geringe Bedeutung. **Die entscheidendere Determinante für die Herzschlagverlangsamung ist die fetale Hypoxie,** die ihrerseits zu einem extremen Anstieg des FA BP und zu einem rapiden Abfall der FHR führt.

Schlüsselwörter: Barorezeptorenantwort, Blutfluß durch die Nabelschnur, fetale Herzfrequenz, fetaler Blutdruck, fetales EEG, Säure-Basen-Gleichgewicht, vollständiger Nabelschnurverschluß.

Résumé

Modifications fetales cardio-vasculaires, métaboliques et fonctionnelles cérébrales à la suite de l'occlusion cordonnale totale.
L'éffet de l'occlusion funiculaire totale (OFT) sur l'oxygénation foetale, sur les paramètres métaboliques et cardio-vasculaires ainsi que sur la fonction cérébrale chez le foetus de mouton a fait l'objet de cette étude.
Nous nous sommes attachés de distinguer les altérations cardiovasculaires à la suite de la OFT dues respectivement à l'hypoxie et aux barorécepteurs.
Les investigations ont porté sur 11 moutons près du terme gravidique dont l'âge de grossesse se situait entre le 126e et le 137e jour. Afin d'enregistrer la tension artérielle foetale (TAF) ainsi que la fréquence cardiaque foetale (FCF) des catheters ont été placés à l'intérieur de l'aorte foetale. Le pH, la P_{CO_2} et la SO_2 ont été déterminés sur d'échantillons de sang obtenus. Une sonde à flux électromagnétique a été installée autour de la veine ombilicale. L'EEG foetal a été enregistré au niveau de l'hémisphère droit. Tous les paramètres ont été étudiés pour des temps variables de OFT.

Résultats:
A la suite de l'OFT la SO_2 artérielle foetale décroit linéairement, s'accompagnant d'une chute du pH, d'une élévation de la P_{CO_2} et suivie d'une acidose métabolique. L'EEG foetal devenait plat après environ 60 secondes d'hypoxie foetale.
La tension sanguine foetale systolique et diastolique s'élève après 0,25 sec.; ceci s'accompagne d'une chute de la FCF.
L'nalyse des modifications de la FCF et de la TAF à la suite immédiate de l'OFT montre une corrélation entre l'augmentation de la TAF et l'équilibre foetal acido-basique.

Conclusions:
Les observations permettent de conclure que la réponse initiale de la FCF à la suite de l'OFT en set une baroréceptrice liée à l'augmentation soudaine de la TAF. Cet état est bref et de faible importance pour l'interprétation de la fréquence cardiaque foetale pendant le travail.
La bradycardie est liée éssentiellement à l'hypoxie foetale entrainant une augmentation extrême de la TAF et la chute progréssive de la FCF.

Mots-clés: Occlusion cordonnale totale, préssion sanguine foetale, fréquence cardiaque foetale, réponse baroréceptrice, équilibre acido-basique, flux sanguin ombilical, EEG foetal.

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Reprint request for Europe:
Prof. Dr. Wolfgang KÜNZEL
Universitäts-Frauenklinik
Josef-Schneider-Str. 4
D-8700 Würzburg/Germany

Reprint requests for the United States:
Leon I. MANN, M.D., Department of Obstetrics and Gynecology, University of Vermont, Burlington, VT 05401